Lixelle ameliorates idiopathic thrombocytopenic purpura

Sir,

Here we report an interesting case of idiopathic thrombocytopenic purpura (ITP). The case was a male patient born in 1925. He was healthy until he manifested epistaxis and proteinuria in 1975. At that time, his platelet count was $0.5 \times 10^4 \text{ mm}^3$. Bone marrow examination revealed normal haematopoiesis with normal megakaryocyte number, consistent with ITP. Anti-DNA antibodies were negative. Renal biopsy was not performed because of bleeding tendency. Steroid was administered without beneficial effects, and tapered later. Although splenectomy was recommended, the patient declined major surgery. Despite conservative treatment, he required maintenance haemodialysis from 1980 (3 days/week). Platelet counts were kept low between 0.5 and $1.5 \times 10^4 \text{ mm}^3$ after the initiation of dialysis therapy. The patient underwent endoscopic surgery for carpal tunnel syndrome in 1999. Since 2000, we have applied lixelle (1.5 m$^2$), a beta2-microglobulin adsorption column (Kanegafuchi Chemicals, Tokyo, Japan), in every dialysis to slow the progression of dialysis-related amyloidosis [1]. Lixelle was set in bloodline prior to the polysulfone dialyser (1.6 m$^2$). Dialysis was performed with a total of 1000 U of heparin for 4 h. Blood and dialysate flows were maintained at 200 and 500 ml/min throughout the haemodialysis-haemoperfusion sessions, respectively. The platelet count gradually increased to $3.5 \times 10^4 \text{ mm}^3$ (Figure 1). The frequency of epistasis decreased markedly. In 2002, the platelet count was $4.2 \times 10^4 \text{ mm}^3$.

The pathogenesis of ITP appears attributable to autoreactive synthesis of autoantibodies to glycoprotein IIb and/or III on platelets [2]. Platelet-associated IgG (PAIgG) was selected as a probe, because it quantifies the activity of ITP [3]. Exposure to lixelle for 20 min reduced PAIgG by 32% (from 310 to 210 ng/ml), but not total IgG (from 1160 to 1120 mg/dl).

Fig. 1. Effects of lixelle on platelet count. Platelets were measured twice in every month. The application of lixelle elevated platelets from $1.1 \pm 0.4$ to $3.4 \pm 0.9 \times 10^4 \text{ mm}^3$ ($P < 0.05$, Student’s $t$-test), when the mean value for 12 months before initiating lixelle was compared with that for a year after initiating lixelle.
This was also true when the values at 20 min of haemodialysis/haemoperfusion sessions (PAIgG, 227 ng/ml; IgG, 1204 mg/dl) were adjusted for changes in plasma volume by correcting for haematocrit change [4]. Thus, lixelle probably captured variable domains of the antiplatelet antibody to platelets. Lixelle consists of cetylamine residue, and adsorbs inflammatory cytokines, endotoxin and digoxin as well as beta2-microglobulin [1,5,6]. The present case warrants further studies, assessing electrochemical similarity between cetylamine and glycoprotein IIb and/or III, and suggests a potential use of lixelle for ITP treatment.

Currently, large doses of immunoglobulin administration and/or platelet transfusion are the treatment of choice for aplastic or lytic crisis of ITP. Although more solid clinical evidence is required to draw a definite conclusion, the present findings suggest that lixelle which does not transmit any blood-borne pathogens may become a new therapeutic alternative for ITP.

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